Amendments to the Specification

The specification has been amended as follows (underlines indicate insertions and ~~strikeouts~~ indicate deletions):

Please replace the title with the following:

THE SCN1A LOCUS ~~LOCI~~ FOR IDIOPATHIC GENERALIZED EPILEPSY, MUTATIONS THEREOF AND METHOD USING SAME ~~TO ASSESS, DIAGNOSE, PROGNOSE OR TREAT EPILEPSY~~.

Please replace the paragraph starting at line 26 at page 6 and ending at line 5, at page 7, with the following amended paragraph:

While human SCN1A, SCN2A and SCN3A are preferred sequences (nucleic acid and proteins) in accordance with the present invention, the invention should not be so limited. Indeed, in view of the significant conservation of these genes throughout evolution, sequences from different species, and preferably mammalian species, could be used in the assays of the present invention. One non-limiting example is the rat SCN1A ortholog gene which shows 95% identity with the human SCN1A gene~~, at the amino acid level.~~ The significant conservation of the mouse SCN1A gene can also be observed in OMIM (see above).

Please replace lines 1 to 12 in page 27, with the following amended paragraphs:

Figure 2 shows the PCR primers used for genomic PCR-SSCP of SCN1A (SEQ ID NOs: 99-188);

Figure 3 shows the sequence of the SCN1A mutations found in epilepsy patients (SEQ ID NOs: 189-192 and 309);

Figure 4 shows the PCR primers used for genomic PCR-SSCP of SCN2A (SEQ ID NOs: 193-306);

Figure 5 shows the mutation found in epilepsy patients in SCN2A (SEQ ID NOs: 307 and 308);

Figure 6 shows the PCR primers used for genomic PCR-SSCP of SCN3A (SEQ ID NOs: 310-399); and

Figure 7 shows the mutation found in epilepsy patients in SCN3A (SEQ ID NOs: 400-408).

Please replace the paragraph starting at line 12 at page 58 and ending at line 15, at page 59, with the following amended paragraph:

One such example of functional studies was investigated by assessing the effects of mutation D188V in the SCN1A gene on sodium channel function by introducing the mutation into a cDNA encoding the rat ortholog SCN1A gene. This rat ~~rate~~ gene shares > 95% identity with the human SCN1A gene~~, at the amino acid level~~. The expression of wild type and mutant channels in *Xenopus* oocytes, and the examination of their properties using voltage-clamp electrophysiological recording is amenable to this *Xenopus* system. Wild type sodium channels are closed at hyperpolarized membrane potentials. In response to membrane depolarization the channels open within a few hundred microseconds, resulting in an inward sodium flux, which is terminated within a few milliseconds by channel inactivation. In whole cell recordings, rapid activation and inactivation of thousands of sodium channels distributed throughout the cell membrane results in a transient inward sodium current that rises rapidly to peak amplitude and then decays to baseline within a few milliseconds. Among the channel properties that are likely to be altered by mutations linked to epilepsy are: 1) the voltage-dependence of activation, a measure of the strength of membrane depolarization necessary to open the channels; 2) the voltage-dependence of steady state inactivation, a measure of the fraction of channels available to open at the resting membrane potential; and 3) the time course of inactivation. Preliminary results indicate that D188V mutant channels are identical to wild type channels with respect to the voltage-dependence of activation and to inactivation time course. However, steady state inactivation for the mutant channels is shifted to membrane potentials that are slightly more positive than observed in wild type channels. This positive shift should increase the fraction of channels available to open at rest. This could increase neuronal excitability and contribute to epileptogenesis. Thus, a functional consequence of a naturally occurring mutation in a sodium channel gene has been tentatively identified. Thus, the functional consequence of the D188M mutant could at least in part explain its role in epilepsy. Such a functional consequence is expected to be observed with other mutations identified above in SCNA1, SCNA2 and SCNA3.